Textbook of Sports Medicine

Basic Science and Clinical Aspects of Sports Injury and Physical Activity

Edited by
Michael Kjær, Michael Krogsgaard
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Harald Roos, Timo Takala
Savio L-Y Woo

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Science

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Preface

In past decades the number of exercising individuals and the area of sports medicine have grown considerably. Sports medicine has developed both in terms of its clinical importance with appropriate diagnosis and adequate rehabilitation following injury as well as its potential role in the promotion of health and prevention of life-style diseases in individuals of all ages. Furthermore, lately the medical field has gained improved understanding of the use of physical activity as a treatment modality in patients with a variety of chronic diseases and in rehabilitation after disabilities, injuries and diseases. Common to these advancements is the fact that a certain amount of clinical experience has to be coupled with sound research findings, both basic and applied, in order to provide the best possible recommendations and treatments for patients and for the population in general.

There is a tradition in Scandinavia for an interaction between exercise physiology and clinical medicine and surgery, and it is apparent that both areas have hypotheses, inspiration and possible solutions to offer each other. It is therefore apparent that a textbook on sports medicine must attempt to incorporate all of these aspects to be comprehensive. A historical or classical reference has been selected as an introduction to each chapter to reflect the impact that a specific scientific work has had on that field. Having several authors collaborating on each chapter in the book ensures both diversity and a degree of consensus in the text, which will hopefully make the book usable as a reference book, and as a textbook both at the pre- and postgraduate levels. It has been our goal to address each topic within sports medicine in a scientific way, highlighting both where knowledge is well supported by research, as well as areas where the scientific support is minimal or completely lacking. It is the intention that the book will help the people who work clinically within the area of sports medicine in their daily practice, and that it will also provide the basis for further research activity within all areas of sports medicine. Moreover, we wish to highlight where knowledge and methodologies from different, and often distant, areas can interact to create a better understanding of, for example, the mechanisms behind development of tissue injury and its healing.

The editorial group has been delighted that some of the world's leading experts have agreed to participate in this project, and they have all contributed with informative and very comprehensive chapters. I greatly appreciate their contribution and that of the editorial group who worked hard on the completion of the book. Additionally, I wish to acknowledge all other contributors who have helped with the practical procedures of this project. Finally, I hope the reader of this book will share the research dreams, the clinical interest, and the enthusiasm in relation to the sports medicine topics with that of the authors and the entire editorial group.

Michael Kjaer
Copenhagen, September 2002

Introduction

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The exercising human: an integrated machine

Physiological boundaries have fascinated man for a long time, and achievements like climbing up to more than 8800 m above sea level without oxygen supply or diving down to more than 150 m in water without special diving equipment are at the limit of what textbook knowledge tells us should be possible for humans. Likewise, athletes continue to set new standards within sports performance, and patients with chronic diseases master physical tasks of a very challenging nature, like marathon running, that hitherto were thought impossible.

Muscles, tendons and bone are elegantly coupled together to provide an efficient system for movement, and together with joint cartilage and ligaments they allow for physical activity of various kinds. In order to provide energy to contracting muscles, ventilation often rises 20–40-fold and cardiac pump function can increase up to 6-fold during strenuous exercise in well-trained individuals in the attempt to deliver sufficient oxygen to allow for relevant oxidative processes that can be initiated within seconds. In addition, working skeletal muscles can by training achieve substantial increases in their capacity to both store energy and to extract and utilize oxygen. With regards to endurance capacity, humans are still left with the fact that the size of the heart relative to the skeletal muscle is relatively small — even in top-class runners — compared to basically all other animal species.

To drive the human machinery, local as well as distant substrate stores provide fuel for energy combustion, allowing for very prolonged exercise bouts. A controlled interplay between exercise intensity, energy metabolism and regulatory hormones takes place, and intake of different food stores can cause the muscle to adjust its fuel combustion to a large degree. The initiation of signals from motor centers to start voluntary movement and afferent signals from contracting muscle interact to achieve this and several signalling pathways for circulatory and metabolic control are now identified. The brain can make the muscles move, and can at the same time use substances for fuel that are released from muscle. Furthermore, intake of different food sources can cause the muscle to adjust its fuel combustion to a large degree.

Training can cause major tissue and organ adaptation and it is well known that this to a large degree depends upon both genetic and trainable factors (Table 1). More recent studies on identical twins have allowed for a discrimination of these two factors in relation to exercise and have shown that between 30 and 50% of the variation in parameters like maximal oxygen uptake or muscle strength are likely to be attributed to genetic factors. Rather than discourage humans from starting training on this background, it is fascinating to identify factors responsible for training improvements in, for example, muscle tissue. It is evident that contractile force can elicit transcription and translation to produce relevant changes in the amount of contractile or mitochondrial proteins, but the underlying mechanism in both muscle and connective

Table 1 The capacity of various tissues and systems, and their ability to adapt to physical activity or inactivity.

Function	Increase during single bout of physical activity ultimate tensile strength	Improvement with training (%)	Time required for adaptation	Decrease in function or maximal load with 3–4 weeks of inactivity (%)
Cardiorespiratory			Months–years	
Ventilation	35-fold	0		
CO	6-fold	90		40
O_2 extraction	2–3-fold	25		30
VO_2	12–18-fold	50–60		40
Muscle metabolism			Weeks–months	
Glycogen/fat stores	–	100		50
Oxidative capacity	–	300		40–100
Connective tissue			Months–years	
Tendon	100 MPa	20		30
Ligament	60–100 MPa	20		30
Bone	50–200 MPa	5–10		30
Cartilage	5–40 MPa	5–10		30
Muscle			Months–years	
Strength	–	100–200		60
Fibre CSA				
Type I	–	40		20
Type II	–	80		30

CO: cardiac output; VO_2: whole body oxygen uptake; CSA: cross-sectional area.

tissue is not understood. Interestingly, substances are now being identified (e.g. mitogenactivated protein kinases) where subtypes are differentially activated by either metabolic stress or by the degree of contractile stress, to cause either increased cell oxidative capacity or muscle cell hypertrophy, respectively. We are therefore at a point where we can begin to master the study of the adaptation of the human body not only to acute exercise, but also to loading and overloading, and this will provide us with prerequisites for study of the ultimate adaptation potential that the human organism achieves, and thereby better describe also on an individual level why tissue becomes overloaded and injured.

The delicate balance between training adaptation and injury—the dilemma of rehabilitation

It is important for the clinician who treats the recreational or elite athlete to have a thorough understanding of the injury, and also the ability of the affected tissue to adapt to immobilization, remobilization and training. One example is the considerable plasticity that skeletal muscle tissue displays. While strength is lost (up to 60%) rapidly within a few weeks of immobilization, it can be regained over the next couple of months, and strength can be augmented up to 2-fold with training for extended periods (months/year). Bone loss also (up to 40%) occurs rapidly within weeks of immobilization and is subsequently regained in the following months of rehabilitation. However, somewhat in contrast to muscle, extended training periods have a relatively modest impact on bone tissue augmentation. Connective tissue loss in tendon is also comparable to muscle and bone; however, in contrast, its slower metabolism requires perhaps up to 12 months or more before complete tissue recovery from an injury and subsequent inactivity. Thus, an injury that demands a limb to be immobilized for a given length of time may require different time periods for the various tissues to return to their preinjury levels.

In this context it is important for the clinician to note that the cardiovascular system recovers the fastest after

a period of relative inactivity, which may create a dilemma: the athlete wants to take the rehabilitation and training program to new and challenging levels, but the different tissues may not be able to withstand the associated loads, and re-injury or a new so-called 'overload injury' may result. Thus, a thorough understanding of how tissues adapt to physical activity or lack thereof is paramount for the effective treatment and rehabilitation of the injured person.

While acute injury during exercise may intuitively be somewhat easy to understand, it may be more challenging to grasp the insidious and frequent 'overuse' injuries that occur with training. Some important observations in the field of sports medicine have been made in recent decades that have improved our understanding of these injuries. An awareness of the subject's loading pattern is important, of course. The recreational athlete who runs 5 km/week may subject each lower limb to approximately 2100 landings and take-offs in that time period. In contrast, the long distance runner who runs 100 km/week may subject each lower limb to approximately 38 000 landings and take-offs. Clearly, a certain degree of appropriate tissue adaptation has already taken place to withstand these vastly different loads, but nevertheless, injuries may be sustained by both the recreational and elite athlete and therefore remains an enigma. Interestingly, the weekly loading of tissue induced by sports participation is equivalent to that established by national authorities as the upper limits for what is tolerable for manual labour, suggesting that perhaps there is an inherent tissue limitation to loading.

Disadvantageous alignment, like severe pes planus or genu valgus, for example, may be important factors in determing who can withstand a given loading pattern, although such internal factors cannot entirely explain overuse injury. It has become generally accepted that it takes appreciable time for tissues like connective tissue to adapt to a new or increasing demand, even for the most genetically fortuitous. Therefore, any desired progression or change in a training program should be gradual. However, more detailed information with respect to the training frequency, duration and intensity that is required to avoid an injury is currently lacking, and thus preventative efforts in this respect remain difficult. At the same time, it is becoming increasingly appreciated that tissues need restitution periods to 'adapt' to the previous bout of physical activity. This is put into practice, for example, by the tri-athlete who loads the cardiovascular system considerably on a daily basis, but stresses the musculo-skeletal system alternately by training either cycling, running or swimming, which may help to avoid injury. It is during the restitution period that tissues are allowed to recover, or further adapt to an increasing demand by either expanding their quantity or improving their quality. It is likely that in years to come researchers will furnish new and improved measurement techniques that will yield important detailed information about tissue adaptation to physical activity and restitution.

Sports injuries and development of treatment: from recreational sports to elite athletes

In many situations the transformation from overload symptoms to a sports injury is poorly defined and understood. Intensified research in anatomy, biochemistry, physiology and mechanisms of tissue adaptation to mechanical loading is needed to provide the basic understanding of overload injury pathogenesis. Although this in itself represents a paramount challenge, it seems even more difficult to understand an individual's disposition for developing symptoms. Why does one individual develop severe Achilles tendon pain in connection with a certain amount of running, while others do not? Why are overhead activities very painful for some athletes but not for others? Why is the functional stability of a cruciate ligament deficient knee or a mechanically unstable ankle joint different between persons despite the same activity level? Obviously it would be essential to identify the weakest link in each individual case, but knowledge of the individual specific factors is very incomplete. Could there be physiologically different levels for initiation of symptoms in different individuals? It is well known that persons with decreased sensory inputs, for example caused by diabetic polyneuropathy, have a high rate of overload injuries like tendonitis or stress fractures, simply because the natural alarm system is out of order. If a physiological difference in, for example, the threshold of sensory inputs exists in otherwise healthy people, the difference between a

mechanical load that causes symptoms and one that results in tissue damage would vary from person to person.

Treatment of sports injuries represents major challenges. First, the aim to reduce symptoms is demanded by the athlete, and several pharmacological treatments will work well at rest, but will not provide pain relief when the individual is exercising. Secondly, when surgical treatment is indicated to repair irreversible changes of tissues (e.g. rupture of anterior and posterior cruciate ligaments of the knee) or to change biomechanical inferior or insufficient movement patterns (e.g. multidirectional instability in the shoulder) the procedures need to be minimally invasive in order to leave the remaining tissue as intact as possible and to allow for a quick regeneration process. Thirdly, the rehabilitation procedures and time allowed for recovery will be challenged. This is because athletes are eager to return to their sports. In this aspect, similarities can be drawn to occupational and rehabilitation medicine, which aims towards getting the patient back to the functional level that is required to perform a certain labour task.

In contrast to the little which is known about the individual-based factors, there is increasing knowledge about injury mechanisms in athletic performance. A number of specific pathological entities have been recognized, especially during the past two decades, e.g. secondary impingement and internal impingement of the shoulder in overhead athletes. On the basis of recognizing certain common patterns of injury and understanding their pathogenesis, specific treatments — surgical as well as nonsurgical — have been developed. Probably the first injury to be recognized as a specific lesion connected to sports performance was the Bankart lesion of the shoulder, described in 1923, and the way to repair the lesion was obvious once the pathoanatomical background was established. Similarly, when the SLAP lesion of the labrum in the shoulder was described for the first time about 10 years ago, the surgical treatment options could be defined (for further details see Chapter 6.5).

Arthroscopy, which was introduced for knee disorders back in 1968 and developed for the treatment of shoulder, elbow and ankle disorders in the 1990s, has made direct visualization of joint movement and intra-articular structures possible, and has increased the understanding of many intra-articular sports injuries. For the individual athlete it has resulted in a much more specific diagnosis and treatment, and consequently rehabilitation has become faster and easier than after open surgery. Furthermore, the invention and development of magnetic resonance imaging in the early 1990s, and the refinement and general availability of ultrasound investigation during the late 1990s, has increased the spectrum of diagnostic tools significantly. What still requires specific attention is the relative use of these para-clinical supplements as compared with a good clinical examination and judgement. There is no doubt that the new 'machine-tools', developed to help the sports medicine practitioner, tend to be 'over-used' in the initial phase, which is often followed by a more balanced phase in which it becomes evident that patient history and clinical examination can never be replaced by para-clinical tools, but that the latter provides a fruitful supplement in the process of diagnosis in sports medicine.

The collection of clinical information on symptomatic conditions in athletes can lead to identification of uniform patterns and logically based treatment modalities. Series of treated patients can also give information about the success rate of certain treatments, whereas only randomized studies can identify the best treatment strategy in a specific condition. Unfortunately, there are very few randomized studies in sports medicine and especially within traumatology. This is often due to a high demand for treatment to ensure fast recovery and return to sports participation, and it is unlikely that more than a small part of the surgical and nonsurgical treatment modalities will ever be evaluated by randomized studies. Even though more than 60 000 anterior cruciate ligament (ACL) reconstructions are performed every year in the USA, it is unknown which treatment strategy is the most advantageous. There are different factors influencing the decision to perform ACL reconstruction: the chance to get back to sports, prevention of secondary meniscus and cartilage injury, prevention of giving-way or subluxation episodes, risk for anterior knee pain or other operative complications, or timing of surgery. There is no evidence for how these factors should be weighted, and it is unknown if routine reconstruction in all patients shortly after an ACL injury would reduce the risk of late complications and increase activity level better

than a more conservative approach with rehabilitation as primary treatment. It is very important to perform randomized trials at the same time as new treatments are introduced, as it is almost impossible to return to such studies later.

Most rehabilitation programs are based on individual, clinical experience and theoretical principles. Just as with surgical treatment, evidence is still lacking on the effect of a number of general treatment principles. Rehabilitation is very costly, and it is desirable with further development of evidence-based rehabilitation strategies.

New technologies will probably influence the treatment of sports injuries in the near future. Local availability of growth factors may reduce repair and remodelling time after injury or surgery. Scaffolds can be used to introduce a specific architecture. These can be taken over by living tissue, and in combination with controlled gene expression, injured tissue can possibly be restored completely. This will contribute to an avoidance of reconstruction with replacement tissue and accompanying suboptimal recovery, as well as ensure the absence of scar tissue otherwise seen in repair.

In the recreational athlete, many overload conditions are often self-limiting. Nature's alarm system works: overloading of tissues often results in symptoms (pain) long before irreversible changes of the tissue structures happen. With a gradual reduction of activity, symptoms and overloading disappears, and the athlete can resume normal activity again. Tennis elbow is a good example of this mechanism. During one season about 50% of middle-aged persons performing recreational racquet sports will experience symptoms of tennis elbow. The majority of these cases resolve without specific treatment. The interesting phenomenon is, why humans often carry on with exercise despite symptoms and signs of overuse. Interestingly, inflammatory reactions within and around tendons are seen in humans and in a few animal species that are forced to run like race-horses, whereas almost all other species (like mouse, rat or rabbit) do not show signs of tendinitis or peritendinitis despite strenuous activity regimens. Elite athletes can be motivated to continue peak performance despite pain or other symptoms, and it can be difficult or impossible for the natural repair processes to take place. Not enough is known about tissue repair and rehabilitation to define the maximum activity in each individual that is compatible with a full and fast repair.

The boundary between trivial, reversible conditions and irreversible, disabling injuries still has to be defined in many sports. As an example, there is an ongoing discussion about the risk for chronic brain damages in boxing. Furthermore, nearly nothing is known about the long-term effect of continued elite sports activity on degenerative changes in the knee after ACL reconstruction. With this lack of evidence about physical consequences of sports injuries, ethical considerations have a central place in advice and planning. The influence of psychological factors such as competition (matches only take up less than 25% of the active playing time in elite handball, more than 90% of the ACL injuries happen there), self-confidence and acceptance of personal limits have to be acknowledged and further knowledge is warranted.

Table 2 Motivation and needs in different individuals with physical training.

	Performance motivation	Disease-effect motivation	Prevention motivation	Guidelines for training	Tolerable amounts of training
Patient	++ (function)	+++	++	+++	+
Recreational sports	+	–	+++	++	++
Elite athletes	+++ (competition)	–	(+)	+++	+++

The motive for performing physical training can primarily be based upon a wish of increased performance either in sports or in everyday life, or be related to a wish of increased health and disease prevention. All three groups of individuals display an individually varying degree of which for achieving mental well-being in relation to exercise. The tolerable amount of training depends on the ability of the body to withstand loading and varies therefore significantly between athletes and patients, whereas both patients and athletes share a large request for specific guidelines in relation to the training they perform.

Regular physical training: benefits and drawbacks

For more than 5000 years, systematic exercise or sports have been carried out worldwide, and one can easily consider the average individual living today as being much more inactive than they were in the past. It is becoming more and more scientifically documented that physical inactivity is a major risk factor for disease and premature death, and that the magnitude of this lies on the level of other risk factors like smoking, obesity or drinking. Studies have uniformly concluded that being active or beginning physical activity even at an advanced age, will positively influence risk factors for development of inactivity-associated diseases. In spite of the fact that acute training is associated with a transient increased risk of cardiac arrest, taken in the population as a group, as well as the costly treatment of sports injuries, socio-economic calculation has found that, for the recreational athlete, these drawbacks are far outweighed by the cost-saving benefits of physical training such as lower incidence of diseases, faster hospital recovery after disease in general, as well as a lower frequency of infection and time away from work due to sickness. The field of sports medicine is therefore facing a major challenge in improving the level of physical activity in the general population, and for setting up overall guidelines.

Physical training and patients with chronic diseases

Acute and chronic diseases are associated with both organ specific manifestations as well as by more general disturbances in function due to physical inactivity and sometimes even additional hormonal and cytokine-related catabolism. In general, physical training can counteract the general functional disturbances, and maybe even affect or prevent the primary manifestations of disease. It is important to note that the motivational aspects, as well as the requirements for supervision and guidelines, in the patient with a present disease differ markedly from healthy exercising individuals (Table 2).

In principle, most diseases can be combined with a certain degree of physical activity, but the amount of restrictions put upon the patient differs considerably between diseases (Table 3). Certain diseases have been shown to be influenced greatly from physical activity

Table 3 Effects of physical training upon different diseases.

Diseases in which physical training will act preventively in disease development and positively upon primary disease manifestations
Ischemic heart disease
Recovery phase of acute myocardial infarction
Hypertension
Type-2 diabetes
Obesity (most pronounced with respect to prevention)
Osteoporosis
Age-related loss of muscle mass (sarcopenia)
Osteoarthritis (most likely only the prevention)
Back pain
Cancer (prevention of colon and breast cancer)
Depression and disturbed sleep pattern
Infectious diseases (prevention of upper respiratory tract infection)

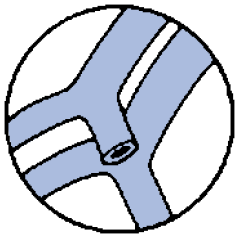
Diseases in which moderate or no direct effect can be demonstrated upon the primary disease manifestations, but where exercise will positively affect both health associated risk factors and the general disturbances in overall body function
Peripheral vascular diseases (arterial insufficiency)
Type-1 diabetes
Bronchial asthma
Chronic obstructive lung disease
Chronic kidney disease
Most forms of cancer
Most acute and chronic liver diseases
Rheumatoid arthritis
Organ transplanted individuals
Spinal cord injured individuals
Most neurological and mental diseases

Diseases in which much caution has to be taken or where exercise is to be discouraged, and where physical training often can have a worsening effect upon primary disease manifestations or may lead to complications
Myocarditis or perimyocarditis
Acute heart conditions (e.g. unstable angina, acute AMI, uncontrolled arrhythmia or third degree AV-block)
Acute infectious diseases associated with fever (e.g. upper respiratory tract infection)
Mononucleosis with manifest splenomegaly
Aorta stenosis (chronic effect)
Acute severe condition of many diseases mentioned above (e.g. severe hypertension, ketoacidosis in diabetes)
Acute episodes of joint swelling (e.g. rheumatoid arthritis) or severe muscle disease (e.g. myositis)

(e.g. ischemic heart disease, type-2 diabetes), whereas other diseases are known to be relatively insensitive to exercise when it comes to primary disease manifestations (e.g. chronic lung disease, type-1 diabetes). In the later group of diseases, it should, however, be noted that physical training can still have a beneficial effect on health-related parameters that can be achieved by individuals in general. This effect is achievable even in the absence of any worsening of the primary chronic disease. This emphasizes the importance of also encouraging individuals with chronic (and not necessarily fatal) diseases to train on a regular basis from a general health perspective. In addition, almost all diseased individuals can exercise in order to counteract the general loss in function that their disease-related inactivity has caused. In very few cases, extreme caution has to be taken when performing exercise (e.g. acute infectious diseases) (Table 3).

In spite of current knowledge of the effect of physical training on diseases, the exact mechanisms behind this are still only partially described. To find such biochemical and physiological pathways will be important not only for addressing which type and dose of physical training should be prescribed for the individual patient, but also for identifying more general 'health-pathways' by which muscular contractions can influence the health status of the individual. Especially in relation to disease, the influence of training on such pathways either by itself or in combination with pharmaceutical drugs will potentially play a role in treatment of disease and maintenance of health into old age. Specific identification of health-related pathways in our genes will furthermore provide insight into the genetic polymorphism and help to explain the interindividual variation in training responses and health-related outcome of these. Evidently, this will also open possibilities for genetic treatment of inherited disorders with regards to tissue and organ adaptability to training, and at the same time inadvertently provide opportunities for misuse of gene therapy in relation to doping, a question that will challenge the sports medicine field ethically.

Part 1
Basic Science of Physical Activity and Sports Injuries: Principles of Training



Chapter 1.1
Cardiovascular and Respiratory Aspects of Exercise — Endurance Training

SIGMUND B. STRØMME, ROBERT BOUSHEL, BJØRN EKBLOM, HEIKKI HUIKURI, MIKKO P. TULPPO & NORMAN L. JONES

Classical reference

Krogh, A, Lindhard, J The regulation of respiration and circulation during the initial stages of muscular work. *J Physiol* (*Lond*) 1913; **47**: 112–136.

This paper demonstrated changes in respiration and heart rate with the transition from rest to bicycle exercise. The investigators did experiments on themselves, and Fig. 1.1.1 shows the changes in tidal air and heart rate at the onset of exercise. As will be noted, a very rapid increase in both ventilation and heart rate was observed, and this led to the conclusion that motor center activity in parallel with activation of skeletal muscle caused an increased stimulation of respiratory centers as well as the heart. This was called cortical irradiation, and has later been referred to as central command or feed-forward, and has become an important topic in the discussion of respiratory, circulatory, and hormonal changes during exercise.

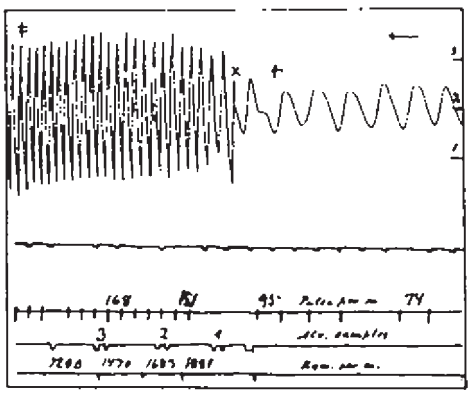


Fig. 1. J. L. Transition from rest to work. Exp. X. Scale in liters. Time in ¼ minutes. + Ready, × Begin, ⊕ Stop.

Fig. 1.1.1 A recording of the tidal air on a spirometer (constructed by Krogh) at rest and at the beginning of exercise.

Cardiovascular adaptation

Cardiac output

The pumping capacity of the heart is a critical determinant of endurance performance in exercise events such as running, cycling, rowing, swimming, etc., where a large fraction of total body muscle mass is contracting dynamically. Because of the large dependence on oxidative metabolism for the total energy turnover in exercise activities sustained for longer than 3 min, performance level is, as will be discussed later, largely dependent on the capacity for O_2 delivery, and thus on the magnitude of maximal cardiac output.

Maximal aerobic power ($\dot{V}O_{2\,max}$) is a classic measure of the capacity to perform endurance exercise, and may be described physiologically as the product of cardiac output and the extraction of O_2 by muscle. For almost a century it has been recognized that a linear relationship exists between maximal oxygen uptake and cardiac output, and this relationship is also observed in other species [1–3]. It is estimated that 70–85% of the interindividual difference in $\dot{V}O_{2\,max}$ is

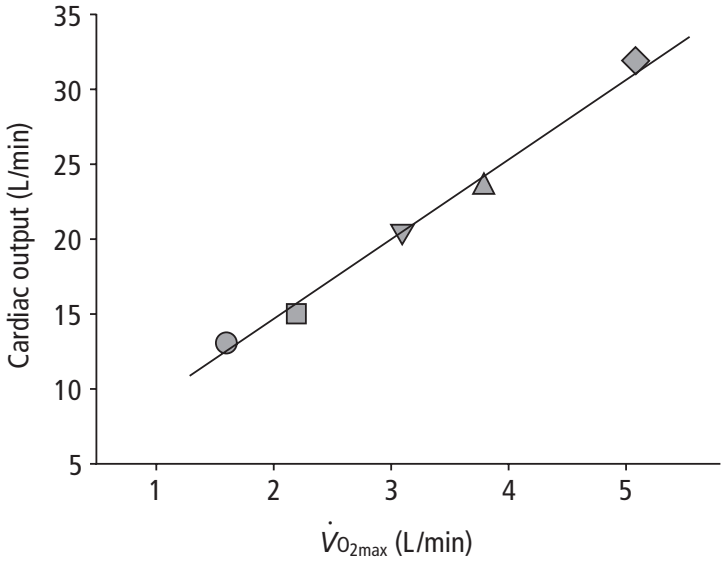


Fig. 1.1.2 Relationship between increases in cardiac output and maximal oxygen uptake in heart failure patients (circles), healthy males after 20 days' bedrest (squares), the same subjects before bedrest (inverted triangle), the same subjects after endurance training (upright triangle), and endurance athletes (diamonds).

attributable to the level of maximal cardiac output [4]. Looked at another way, during whole body exercise, only ~60–80% of maximal mitochondrial respiratory capacity is exploited because of the limits of O_2 delivery [5–7]. Endurance training augments skeletal muscle oxidative capacity and O_2 extraction, but the principal variant for improvements in $\dot{V}O_{2\,max}$ is maximal cardiac output [8–10] (Fig. 1.1.2).

On the other hand, differences in athletic performance amongst competitive athletes with similar $\dot{V}O_{2\,max}$ are linked to peripheral mechanisms [11], such as running economy. The basic question as to what limits maximal aerobic power ($\dot{V}O_{2\,max}$) will be discussed later in this chapter.

Cardiac structure

The increase in maximal cardiac output (Q_{max}) following endurance training results from a larger cardiac stroke volume (SV), whereas maximal heart rate (HR_{max}) is unchanged or even slightly reduced. While heart size is a function of total body size as well as genetic factors, the higher SV achieved by endurance training is attributed to enlargement of cardiac chamber size and to expansion of total blood volume [12]. On the basis of cross-sectional studies in both female and male endurance-trained athletes, total heart volume is generally 15–25% larger than sedentary size-matched controls, with morphologic differences seen in both the ventricles and the atria [13]. Chamber enlargement is also observed in endurance-trained paraplegics compared to sedentary matched controls [14].

There is a close relationship between cardiac volume and physical performance [12]. However, the cardiac hypertrophy is dependent on the type of sport carried out. There are two main types of myocardial hypertrophy. In weight lifters and other strength-training athletes heart wall thickness is increased, with only minor increases in heart cavity diameters, while endurance athletes have increased heart volume and cavity diameter with a proportional increase in wall muscle thickness [15]. The ratio of wall thickness to cavity diameter is unchanged in the endurance-trained individual but increased as a result of strength training [16].

The left ventricular hypertrophy in the endurance-trained individual is due to volume overload ('eccentric' hypertrophy), while the hypertrophy due to strength training develops as a consequence of pressure overload ('concentric' hypertrophy). Rowing, for